

As Dr Checkley points out, a further stimulus to a continued effort to evaluate this interrelationship comes from the established knowledge that lithium, one of the most effective anti-manic agents, does have a very marked anti-thyroid effect.

We have recently begun to study in a prospective longitudinal design an individual who has both rapidly cycling manic-depressive illness and also severe hypothyroidism when on lithium carbonate. The initial evidence suggests that the thyroid hormones may be capable of playing an important physiological role in the modulation of affective illness, a rising level of thyroid hormone available to the cell being adaptive and therapeutic during depression but maladaptive and capable of inducing mania when bipolar illness is also present.

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SWEAT LITHIUM IN MANIC-DEPRESSION

DEAR SIR,

Miller *et al* in their letter (*Journal*, November 1978, **133**, 477-8) present interesting data on the concentration of lithium in pilocarpine-stimulated sweat,

and go on to suggest that in hot weather some patients may need extra lithium. In most patients, however, due to a concomitant 10-20 fold loss of sodium in sweat, there will be a reduction in renal excretion of lithium with a resultant net gain.

Measurement of electrolyte losses via skin over 24-hour periods is a cumbersome procedure. It was carried out in a 53-year-old female who was receiving a diet containing 95 mEq sodium and 80 mEq potassium, and on a daily dose of lithium carbonate 500 mgm t.d.s. (40 mEq) her serum lithium level was 1.0 mEq/L. During the 1st 3 days of the study, while she was on bed rest at a room temperature of 21-23°C (70-74°F), her daily skin losses were: sodium 0.80, 0.72 and 1.12 mEq; potassium 0.43, 0.50 and 0.70 mEq; and lithium 0.06, 0.06 and 0.07 mEq respectively. On the 4th and 5th day, the room temperature was raised to 30-35°C (86-95°F) while she was still on bed rest, the losses were: sodium 31.44 and 16.68 mEq; potassium 6.13 and 7.76 mEq; and lithium 1.43 and 1.22 mEq respectively (Saran and Russell, 1976). Thus, the amount of lithium lost via skin in hot environment was still too small to make any impact on the daily dose. In contrast, there was a marked loss of sodium.

As Miller *et al* point out, their data is not directly applicable to a clinical situation, but if one were to indulge in a theoretical exercise a patient with a serum lithium level of 1.0 mEq/L would have to lose about 3 litres of fluid as sweat per day to lose 6.9 mEq lithium (equivalent to about 250 mgm lithium carbonate), and this 3 litres of sweat would probably contain 200 mEq sodium (an estimate, as Miller *et al* do not provide figures for sodium concentration). A reduction in renal excretion of sodium by 200 mEq is likely to reduce renal lithium excretion by 50 per cent (Thomsen and Schou, 1968), which depending upon the daily dose of lithium will be in the range of 15-20 mEq. In this hypothetical patient, if sodium and lithium intake remain unchanged, lithium will accumulate at the rate of 8-13 mEq/day.

To summarize, in conditions which lead to excessive sweating there is a large sodium loss, which must be taken into account and either the dose of lithium reduced or the patient advised to increase his sodium intake.

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